

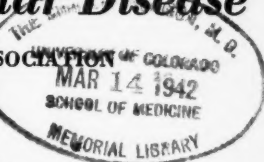
Modern Concepts of Cardiovascular Disease

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TRAUMA AND HEART DISEASE

While the relationship between trauma and heart disease presents, during peace time, many perplexing problems, the advent of war vastly increases the possibility of damage to the heart, both in actual combat and in the preparation under pressure, a necessary part of concentrated war effort. In this discussion the term "trauma" will be used in its broadest sense to include, not only the effects of wounds or blows, but also changes in the heart induced by excessive strain. Trauma may cause structural damage but it may also affect the proper functioning even when there has been no direct injury to the heart. That the heart has been damaged may be perfectly obvious, as in penetrating wounds of the thorax over the cardiac area in which the wound enters one of the cardiac chambers. However, it is possible for the heart to be injured even when there is no evidence of damage to the chest wall. It frequently requires the most detailed examination and a critical evaluation of all the facts before one can determine whether the heart has been involved.

In studying the relationship of trauma to the heart certain fundamental facts must be considered: (1) The condition of the patient prior to the injury. A diseased heart is more likely to be damaged than a normal one. (2) The severity of the injury and particularly the part of the body injured. (3) The type of injury,—a penetrating wound over the precordium would most probably involve the heart. (4) The symptoms present at the time of the injury and shortly thereafter,—whether they were of a cardiac nature or not. Should characteristic symptoms of heart disease be found, it is most important to ascertain the time of their appearance. (5) A thorough examination to determine whether the findings which were attributed to the injury had not pre-existed. (6) Where there is evidence of pre-existing heart disease, the question to be settled is whether this has been aggravated by the injury. (7) Where excessive strain is claimed to be the causative factor, it is necessary to know precisely what was being done at the time of the supposed strain. It is essential to have evidence of activity of a much more strenuous nature than the individual is in the habit of performing normally. The cardiac symptoms and signs must appear immediately, or within a reasonably short time, before causal relationship is established. (8) The effects of extra-

cardiac complications on the heart must be assessed.

(9) The type of individual must be taken into account as those of a nervous disposition are more likely to have many complaints. The possibility of malingering must also be kept in mind. (10) The final result is frequently affected by the treatment given both at the time of the injury and thereafter. Undoubtedly, skilled treatment can, in many instances, materially reduce the period of disability.

Most cardiologists would agree with White that hard physical labor, strenuous athletic sports, repeated trauma of lesser degree, or mild poisoning, have never been proven to be the cause of myocardial disease. One might also state that damage to the normal heart by trauma is uncommon and that, excluding penetrating wounds, no severe, permanent damage to the normal heart takes place except in rare instances. Rheumatic, luetic, hypertensive or arterio-sclerotic heart disease—the common types—are not caused by injury. When these are found the only problem is whether the disease has been aggravated by the injury.

Some of the injuries which can occur to the heart and large blood vessels as a result of trauma will now be considered:

Functional Disorders: There is no doubt that congestive heart failure can be precipitated by trauma in a diseased heart which was previously well compensated. Heart failure attributable to accident should appear within a short time of the injury except in the case of the development of a rapid rate due to paroxysmal tachycardia, auricular fibrillation or auricular flutter, rupture of a valve, or during the course of serious complications following the injury. Very severe damage to the chest can cause acute dilatation and severe heart failure, even in a normal heart. Here the time interval for the onset of symptoms and signs is very short.

Disorders of Rhythm: Premature contractions may appear after an injury but are usually of little significance. Supra-ventricular tachycardia can cause temporary embarrassment but the attack is usually of short duration, either stopping spontaneously or being terminated by appropriate therapy. Ventricular tachycardia is a serious condition and may pass into ventricular fibrillation which is the common cause of sudden death. Auricular fibrillation or auricular

flutter may appear and while the rate is rapid, cause serious impairment of the circulation. Frequently the irregularity ceases spontaneously or normal rhythm can be restored by suitable treatment. Where the irregularity persists, the rate can be controlled by digitalis so that adequate cardiac function may be maintained, although the heaviest forms of physical effort should be avoided. Heart block rarely follows trauma and when it does, indicates hemorrhage into the septum. The block may be only temporary, being caused by the reaction around the area which has been permanently damaged. In older individuals, the common cause of heart block is coronary arterio-sclerosis, so that the decision as to whether the block was due to injury would be facilitated if the patient had a normal electrocardiogram shortly before the accident. It must not be overlooked that in complete heart block an Adams-Stokes attack may be the precipitating cause of the accident.

Pericardium: Pericarditis of some degree always follows penetrating wounds and it may result from a blow to the chest. Non-suppurative pericarditis usually disappears without any important sequelae but suppurative pericarditis is extremely serious and usually ends fatally. Hemopericardium may result from a wound penetrating a heart chamber, hemorrhage from an injured coronary artery, or from cardiac rupture secondary to myocardial damage. As a rule, unless the hemorrhage can be stopped within a very short time, death ensues.

Myocardium: Penetrating wounds may enter the heart chambers and cause immediate death or evidence of hemopericardium with falling arterial and rising venous pressures. Bullets have been reported in the heart cavities in patients who have recovered and in some instances have passed into the peripheral circulation causing embolic phenomena. Similarly, bullets may lodge in the myocardium and cause little apparent effect. If these foreign bodies cause no particular disturbance, it is probably unwise to attempt to remove them. Fracture of the ribs or sternum may damage the myocardium and may even cause a wound which enters one of the chambers. Compression between the sternum and vertebrae can seriously injure the heart and may even cause rupture of the normal heart. Bright and Beck and Schlomka investigated the effects of non-penetrating blows on the heart. They found that the changes depended on the severity of the blow and consisted of disorders of rhythm, a fall of arterial pressure, a rise of venous pressure, a tic-tac quality of the heart sounds, dilatation of the heart, and changes in the electrocardiogram indicating myocardial damage, in some being similar to those seen after coronary occlusion. Most of the deviations from normal disappeared in about a month. In the experimental animals which were killed, the lesions were found to be well healed. When death occurred it followed cardiac rupture, either immediately or during the period that softening of the myocardium was present. The reported cases of cardiac rupture

indicate that any of the chambers may be affected, the incidence being about the same for all. Cardiac aneurysm may also follow severe trauma to the myocardium. In order to establish a relationship between the injury and the cardiac symptoms and signs, these should appear immediately, or within a short time. Despite the fact that such severe effects can follow non-penetrating blows, even in some instances when there is no external evidence of trauma, mild contusions heal without any evidence of permanent impairment of the heart. Indeed, it is remarkable how well the functional capacity is restored after rather severe damage. Anyone who has done cardiac experimental work recognizes the surprising amount of trauma which the heart can withstand without apparently interfering, to any significant extent, with its ability to maintain a normal circulation. In man, the most striking example of this is the satisfactory results which Beck has obtained by means of the operation which he has devised for the treatment of angina pectoris, in which the heart is purposely traumatised in order to create adhesions. The extraordinary thing is not as might be suspected, how little, but how much the heart can stand.

Heart Valves: Cases have been reported in which rupture of a valve has followed an injury. As a rule, valves which rupture have been damaged previously since, in only rare instances, has an authentic instance of the rupture of a normal valve been described. The aortic valve is the one most commonly involved and next in frequency, the mitral. In the latter the chordae tendinae are usually the part torn. There is no evidence that stenotic lesions can be caused by trauma. The diagnosis of rupture of a valve is made by the appearance immediately of a murmur which had not been present previously, or shortly after the injury of cardiac symptoms accompanied by the signs of progressive enlargement of the chamber affected by regurgitation of the blood. It is of paramount importance to be certain, if possible, that the murmur was not present before and functional murmurs must be carefully differentiated.

Acute bacterial endocarditis may follow an injury which has caused septicemia. Subacute bacterial endocarditis presents a difficult problem. The onset of the disease is insidious and in the early stages the patient leads his usual life. Even if an injury is sustained and the characteristic signs of the disease appear later, this does not prove that the injury was responsible. The usual organism associated with subacute bacterial endocarditis is the streptococcus viridans. This is a very uncommon contaminant of wounds except those following tooth extraction. Unless the organism cultured from the blood can also be cultured from the wound or is likely to have infected it, the discovery of subacute bacterial endocarditis at the time of an injury or shortly thereafter is most probably coincidental.

Coronary Artery Disease: The relationship between trauma and coronary artery disease is one of the

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Coronary Artery Disease: The relationship between trauma and coronary artery disease is one of the

most difficult and controversial problems which have to be decided in this field. Only after a thorough study of all the available facts and findings can a reasonable conclusion be arrived at.

Angina Pectoris: This symptom complex is usually caused by athero-sclerosis of the coronary vessels. The first point to be decided is whether the patient really has angina pectoris as there are many causes of pain in the chest besides disease of the coronary arteries. Many individuals are totally incapacitated through the erroneous diagnosis of angina pectoris when careful study shows that the heart is not affected in any way. The physician must have a clear understanding of what constitutes angina pectoris and ascertain that the complaints conform accurately to the symptoms characteristic of myocardial ischemia. If this is done fewer errors will be committed.

Trauma commonly initiates an attack in an individual who has been subject to them before and the attack may be of great severity. Cases have been reported in which the first attack followed an injury but in a large percentage at least the symptoms did not accurately correspond to those of angina pectoris. In order to establish a possible relationship, it is essential that the attacks should appear immediately after the injury. There is no evidence that trauma can cause angina pectoris in an individual with normal coronary arteries. There is also no reason to believe that trauma plays any part in the production of athero-sclerosis which is the usual etiological factor in angina pectoris. As a result of recent studies on vascularization of the arterial wall it has been argued that trauma might cause rupture of the capillaries in the vessel wall, thus inducing intimal hemorrhage which narrowed the lumen of the vessel sufficiently to cause myocardial ischemia, or rupture of a plaque producing thrombosis. However, it has not been proven that rupture of intimal vessels is due to increased coronary pressure, nor has it been shown that such has taken place after an accident. Although angina pectoris may first appear during some unusual effort, there is no evidence at present that the latter plays any part in causing the underlying pathology.

Coronary Occlusion: If an attack occurs at work the relationship to effort or trauma is often raised. There is no satisfactory proof that coronary occlusion can be caused in a normal coronary artery as a result of effort or trauma. The usual underlying cause is coronary athero-sclerosis and the infarct follows

a thrombus in the vessel or prolonged spasm. When an infarct is present but no thrombus can be found on autopsy, it is presumed to be due to the latter. There is little doubt that trauma is not a common cause of the onset of coronary occlusion. The vast majority of attacks come on either at rest or while the individual is performing his usual activities. It would seem essential, therefore, for some unusually severe activity to have been undertaken before trauma can be considered as the precipitating cause of the attack. If this were not so everyone who had an attack while at work should be compensated even if his occupation were sedentary. However, if there is definite evidence that the individual, while undertaking some severe strain to which he was unaccustomed, developed a coronary occlusion, then it would seem justifiable to conclude that the attack was precipitated by the unusual effort. Most probably the occlusion would have developed within a short time in any case and the strain merely hastened matters. In order to establish a causal relationship, the symptoms and signs should appear immediately after the strain or at least within a very short period. If this is not so the onset should be attributed to natural causes. After recovery from a coronary occlusion there is no reason to consider that the individual is totally disabled for the rest of his life. It is undesirable for him to undertake heavy physical labor but there is no evidence that light work is harmful and it is possible for a large majority to undertake this successfully.

Great Vessels: Penetrating wounds may puncture the aorta or pulmonary artery and are quickly fatal. Rupture of the normal aorta following non-penetrating blows is very rare but in the diseased aorta a dissecting aneurysm may follow. The basic cause of this condition is marked athero-sclerosis with hypertension. The onset of dissecting aneurysm is usually accompanied by severe symptoms and these should appear at the time the injury takes place before an association can be established. Following trauma an aortic aneurysm may rupture and is immediately fatal. The pulmonary artery and great veins are seldom injured except by penetrating wounds.

In conclusion, one may state that lesions of the heart and great vessels following trauma are not common. In some cases the connection may be obvious but in other instances the most searching investigation is required before a satisfactory solution is forthcoming.

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